Time course of 'set'-related changes in muscle responses to stance perturbation in humans

Marco Schieppati *†‡ and Antonio Nardone †

*Institute of Human Physiology, University of Genoa, I-16132 Genoa and †‡Posture and Movement Laboratory, Division of Physical Therapy and Rehabilitation, Medical Centre of Rehabilitation, 'Clinica del Lavoro' Foundation, IRCCS, I-28010 Veruno (NO), Italy

1. In standing subjects, toe-down rotation of a supporting platform elicits a medium-latency response (MLR) in tibialis anterior (TA) muscle and a long-latency response (LLR) in soleus (Sol). Toe-up rotation induces a short-latency response (SLR) in Sol and a LLR in TA. When subjects steadily hold onto a stable frame, all responses are decreased, except Sol SLR. The aim of this investigation was to assess whether the response modulation is dependent on information from the hand touching the frame, or whether it anticipates the holding task.

2. The time course of the changes in response amplitude was studied in a time interval centred around the act of holding, performed in a reaction-time mode. Subjects kept their extended arm close to the frame in front of them and brought the hand in contact with the frame in response to a visual go-signal. The platform was moved at different intervals prior to or after the go-signal. Surface EMGs of Sol, TA and deltoid (Delt) were recorded.

3. TA MLR began to decrease when the platform was displaced at an interval of 140 ms after the go-signal, about 200 ms before subjects touched the frame and 120 ms before termination of Delt EMG. Four hundred milliseconds after the go-signal the response reached and maintained maximal inhibition, similar to that occurring under the stationary holding condition. The time course of inhibition of Sol LLR and TA LLR was similar to that of TA MLR, except that LLRs began to decrease at an earlier interval. Due to the different response latency from the onset of the perturbations, the beginning of inhibition of both MLRs and LLRs occurred almost simultaneously.

4. The changes in amplitude of leg muscle responses are not triggered by the go-signal, contact with the frame, or arm motion, suggesting that the modulation is related to the transition to a new, stabilized postural 'set'. The similar extent and parallel time course of MLR and LLR suppression, possibly transmitted through different pathways, points to the spinal cord as the site of action. The lack of depression of the monosynaptic SLR suggests an effect at premotoneuronal level. On the basis of selectivity, latency and time course of the effect, we favour the hypothesis that a monoaminergic pathway from the brainstem is involved.

A sudden displacement of a platform upon which a subject is standing perturbs upright stance, leading to compensatory responses in the leg muscles (Fig. 1*A*). A stretch of the triceps is induced when the platform is rotated in a toe-up direction (upward tilt), and a stretch of the tibialis anterior is induced by a toe-down rotation (downward tilt). The former displacement evokes a short- and a medium-latency response in the soleus and a long-latency response in the antagonist tibialis anterior; the latter displacement evokes a medium-latency response in the tibialis and a long-latency response in the soleus. It is well established that the short-latency responses are mediated by the group Ia spindle afferent fibres. The origin of the medium-latency response remains controversial (Schieppati, 1991): there are indications that it is mediated by spindle group II afferent fibres (Siliotto, Grasso, Nardone & Schieppati, 1995; see Dietz, 1992), even if evidence for a group Ia contribution has been presented (Fellows, Dömges, Töpper, Thilmann & Noth, 1993). As to the long-latency response, it has been hypothesized that it represents a long-loop reflex originating

‡ To whom correspondence should be sent at this address.

in the spindle Ia afferent fibres and travelling through supraspinal pathways (Huttunen & Hömberg, 1990). However, it might well be an autogenetic reflex initiated by impulses evoked in group II fibres, not directly by the platform movement but by the stretch of the muscle occurring during the following body sway (Berger, Dietz & Horstmann, 1988; Scholz, Dichgans, Guschlbauer & Ackerman, 1990).

In previous studies we observed a significant decrease in the amplitude of both medium- and long-latency responses to stance perturbations whilst subjects were holding onto a stable frame instead of standing freely. Those findings were obtained under stationary conditions, i.e. with the subjects holding onto the frame prior to, during and after the perturbation (Nardone, Corrà & Schieppati, 1990*a*; Nardone, Giordano, Corrà & Schieppati, 1990*b*). Therefore, we could not assess (1) whether the response attenuation depended on a new stabilized position or on the new 'external reference', both dependent in turn on the somatosensory information from the contact of the hand with the support, or (2) whether the attenuation was a necessary counterpart of the 'intention' to hold onto the frame, to become more stable.

In this study we attempted to define the time course of the attenuation of response amplitude in the time period centred around the moment when the subject made contact with the frame, thereby passing from a free standing to a holding condition. We expected that analysis of the evolution in time and distribution in space of this effect would provide insight into the underlying physiological mechanisms. Therefore, the experiments were devised to answer the following questions. (1) Is there a consistent time relationship between taking hold of the support frame and the attenuation of the responses? (2) Is this decrease abrupt or gradual? (3) Do all the responses to the perturbations decrease over the same time course, regardless of their latency and the muscle in which they appear? Some of the results have been presented recently in abstract form (Schieppati, Nardone, Grasso & Siliotto, 1993).

METHODS

Seven healthy subjects (3 males and 4 females, aged 23–44 years) were selected for testing. The local ethics committee approved the use of the experimental procedures and subjects gave informed, written consent. During the trials, subjects were asked to stand upright on a movable platform with their eyes open and feet 10 cm apart.

Recording

Surface EMG electrodes, in which integrated differential amplifiers (× 2000) were lodged, were positioned on the posterior lower third (soleus, Sol) and the anterior upper third (tibialis anterior, TA) of the leg, and on the anterior aspect of the shoulder (deltoid, Delt), on the right side of the body. The distance between the two leads of each device was 1 cm. EMG signals were band-pass filtered (30–500 Hz), postamplified (× 25), converted analog-to-digital at a sampling rate of 500 Hz and fed to a PC, together with the platform position signal. Subjects wore a latex glove with an aluminium sheet stuck on to the volar surface. When hand contact was made with the conductive frame, a circuit was closed and a 9 V DC signal was recorded. The time of acquisition lasted 800 ms.

Postural perturbations

Platform movements consisted of toe-up rotations (upward tilt) or toe-down rotations (downward tilt). Velocity of platform movement was 50 deg s^{-1}. At the end of the movement the platform stopped at +3 or −3 deg from its original position, respectively. The time interval between each trial varied randomly from 10 to 15 s.

Procedure

Subjects were instructed to maintain their extended right arm about 2–3 cm above a horizontal stable frame positioned at arm's length in front of them and to take hold of it as quickly as possible in response to a visual go-signal. They simply lowered the arm until the hand rested on the bar, without forcefully grasping on it, and held this position for a few seconds, until the platform was returned to its initial position. Each trial began with the switching on of a red light-emitting diode (LED, go-signal) located about 1 m in front of the subjects, at eye level, in a dimly lit room. The current passed through the LED was five times that necessary to light it to visual threshold. Before each trial, the subject was given a verbal warning signal 3–6 s in advance of the go-signal.

Postural perturbations were delivered in a conditioning-test mode. The go-signal provided the conditioning stimulus and the test stimulus was the platform rotation, so that leg muscle responses became conditioned by the holding task. The platform was moved at random intervals prior to or after the go-signal. Figure 1*B* shows a recording during a typical trial, in which the postural stimulus consisted of a toe-down rotation.

Each of the seven subjects performed between sixty and eighty test trials with random presentation of either type of perturbation. The conditioning-test interval varied from −100 to 500 ms (negative intervals indicate that platform displacement preceded the go-signal). For the sake of representation and analysis, the full range of conditioning-test intervals was divided into classes of 50 ms. For example, the average amplitude of the responses to the perturbations delivered in the −25 to +25 ms period was set at 0 ms (go-stimulus and platform movement starting simultaneously), and so forth.

Data analysis

For each recorded trace, EMG activity was measured off-line by means of a cursor on the computer screen (1 sample = 2 ms). The interval from the go-signal to the end of tonic activity of deltoid EMG was the reaction time (RT). The latency of the responses in the soleus and tibialis anterior was computed as the interval between the onset of platform movement and the beginning of EMG activity. After digital rectification of the raw signal, the area of EMG envelope between the onset and end of the responses was computed. In the case of the long-latency responses (LLRs), a window was chosen which lasted 80 ms from the onset of the responses.

In all subjects, at least twenty randomly distributed trials for each type of platform perturbation without the go-signal (unconditioned stimuli) were delivered during the session. The verbal warning

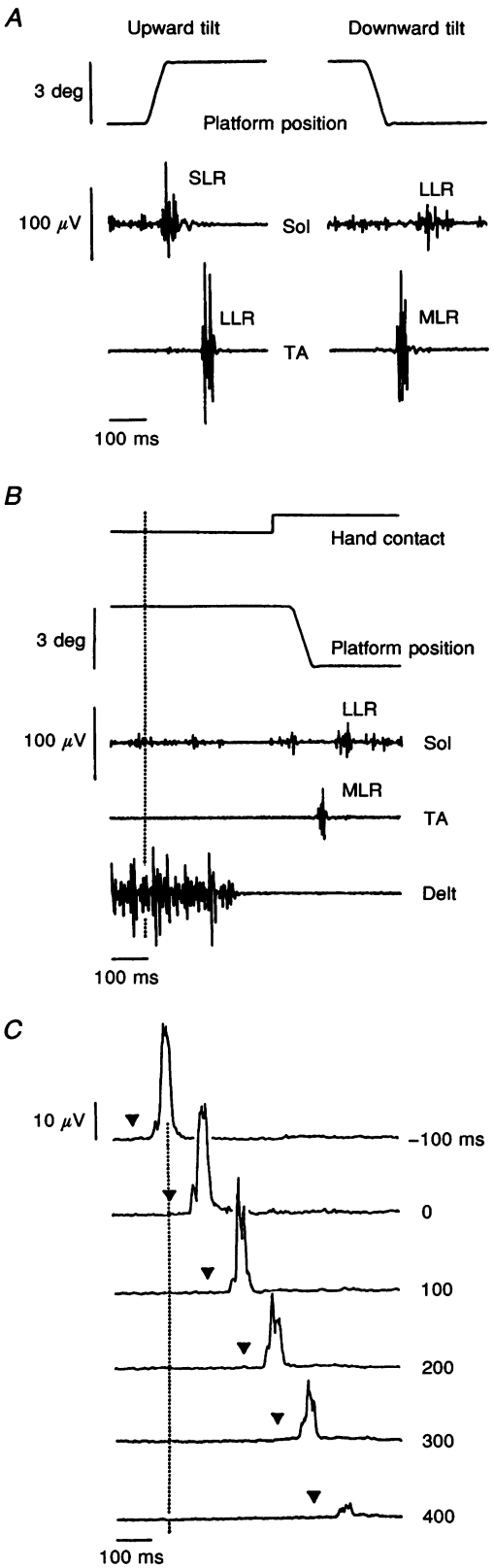

Figure 1. Examples of the EMG responses of soleus (Sol) and tibialis anterior (TA) muscles to platform displacement, recorded under free stance and during the holding task

A, responses to upward and downward tilts. SLR, short-latency response; MLR, medium-latency response; LLR, long-latency response. *B*, display of the signals recorded during a typical holding experiment in the same subject as above. The onset of the visual go-signal (vertical dotted line) was the conditioning stimulus, in response to which the subject made contact with the bar. The task was performed by silencing the tonically active deltoid (Delt) muscle. The test stimulus was downward tilt of the platform. Soon after hand contact, the TA MLR was markedly decreased (compare with Fig. 1*A*, right panel). The Sol LLR was also decreased, although to a lesser extent. *C*, time course of the changes in amplitude of the TA MLR to downward tilt during the holding task. Each trace is the average of 5 rectified and filtered (time constant, 2 ms) EMG signals obtained at different intervals between the onset of platform displacement (▼) and the delivery of the visual go-signal (vertical dotted line). A decrease in the amplitude of the TA MLR already appeared when the platform was moved 200 ms after the go-signal.

signal was also given, as during the conditioned trials. Under this condition (which involved no holding), the area of each response in the leg muscles, averaged through all trials, was taken as the control value. Then, changes in the area of the conditioned responses evoked during the holding task were expressed as a percentage of the control value.

We also acknowledged that subjects could well react to the platform movement instead of the visual stimulus, and that this error might go unnoticed if the platform tilt just anticipated or followed the go-signal. This would not affect the amplitude of leg muscle reflex responses to the platform movement which took place well before hand contact was made. However, since visual RTs were longer than proprioceptive RTs, the error would lead to an underestimation of the 'real' visual RT of the deltoid relaxation. To ensure the detection of such an event, subjects were asked to perform the holding task in response to the go-signal as usual, except that no stance-perturbing stimulus was delivered. Fifty trials were performed and the RTs of the end of deltoid activity obtained under this condition were considered as the deltoid RTs to the visual stimulus. As a consequence, when the difference between the go-signal and the end of deltoid activity was 1 S.D. shorter than the average RT obtained when no platform perturbation was delivered, the trial was rejected. This occurred in 16% of the trials, on average.

Statistical analysis was performed by means of the one-way analysis of variance, and $P < 0.05$ was considered significant. Scheffe's test was used for post-test comparisons. The results of this analysis were not different from those obtained when the mean amplitude of the responses occurring within a given time interval after the go-signal producing the holding task was compared by means of Student's t test to the mean amplitude of the responses occurring prior to the go-signal (see Armitage, 1971).

RESULTS

Leg muscle EMG responses and their modulation by steady holding

An example of the responses evoked by upward tilt and downward tilt is reported in Fig. 1*A*. Upward tilt evoked a short-latency response (SLR) in the soleus muscle, and a long-latency response (LLR) in the tibialis anterior. Downward tilt evoked a medium-latency response (MLR) in the tibialis, followed by a LLR in the antagonist soleus. In accordance with previous findings (Schieppati & Nardone, 1991), the tibialis anterior MLR and LLR were much decreased (to less than 30% of control amplitude) while subjects were holding onto the support frame under stationary conditions. The soleus LLR was also significantly attenuated, although to a lesser extent. The soleus SLR was unaffected. The modulation of the responses obtained during steady holding is reported in Figs 2–4 (filled symbols on the rightmost side of each figure). Hereafter follows a description of the behaviour of the tibialis MLR evoked by downward tilt of the platform during the transition from free stance to holding (an example of which is depicted in Fig. 1*B*), and a brief comment on any similarities or differences between this and the other responses.

Time course of the response modulation

Tibialis anterior MLR. Figure 1*C* shows a series of responses evoked at various conditioning-test delays in one representative subject. The tibialis MLR evoked at the 200 ms interval following the go-stimulus, when the subject was required to perform the holding task, was markedly decreased with respect to those evoked prior to or immediately after the go-stimulus. Such changes in amplitude were not accompanied by obvious changes in latency. When the go-signals were not followed by holding, deliberately or due to subject's inattention, no inhibition of the perturbation-evoked response ensued. Since all the subjects behaved in a similar way during the holding task, with respect to both timing and degree of amplitude reduction, results have been expressed as grand means obtained by pooling the data from all subjects.

Figure 2*A* shows the time course of the changes in the area of this response, averaged from all subjects. The MLRs evoked by perturbations delivered within a period ranging from the 100 ms interval prior to the go-signal until the 150 ms interval following the go-signal were not different from the responses evoked in the absence of the go-signal (controls, continuous line set at 100%). At the 200 ms interval, the response was significantly decreased with respect to controls. The responses evoked afterwards were even smaller, and the minimum value was reached at about 400 ms after the go-stimulus. This amplitude, in turn, was not different from that of the responses evoked during stationary holding (filled circle on the right). Similar plots were obtained for each single subject, but in individual subjects the value of the significance test was affected by the relatively limited number of responses occurring in some of the intervals. On average, but this was also true for all subjects, the amplitude of the responses began to significantly decrease before the hand had come into contact with the bar (this time is indicated by the filled triangle), and while the deltoid muscle was still active (the open triangle indicates the termination of deltoid EMG). The 'true' time at which the response began decreasing is hard to tell, due to the grouping of data within intervals of 50 ms duration: a safe assumption would be to equate the onset of response attenuation with the intercept of the curve (3rd order polynomial) best-fitting the data points with the horizontal dotted line drawn 1 S.D. below the control value. In this case, the attenuation process would begin to affect the responses evoked by platform displacements starting from about 140 ms after the go-signal.

Figure 2*B* shows the changes in amplitude of the tibialis anterior MLR replotted as a function of the end of deltoid activity, i.e. 245 ms from the go-signal (the mean RT of

EMG termination, now made equal to the origin of the abscissa). The deltoid signal was back-averaged, after rectification, with respect to the time of hand contact, and its profile reported as an inset in the graph, on the same time scale (note, however, that the EMG trace reaches the baseline with some delay with respect to 0 ms, since EMG termination at time 0 was the statistical mean, whilst the electronic average was influenced by the trials with a somewhat later full muscle relaxation). With the assumption that a close association existed between response depression and execution of the holding task, it was hypothesized that the termination of EMG activity was a more precise time reference than the go-signal, being free from the subjects' fluctuations of attention. However, the slopes of the best-fit curves drawn through the data points in both cases (*A* and *B*) were very similar. The only obvious changes with the

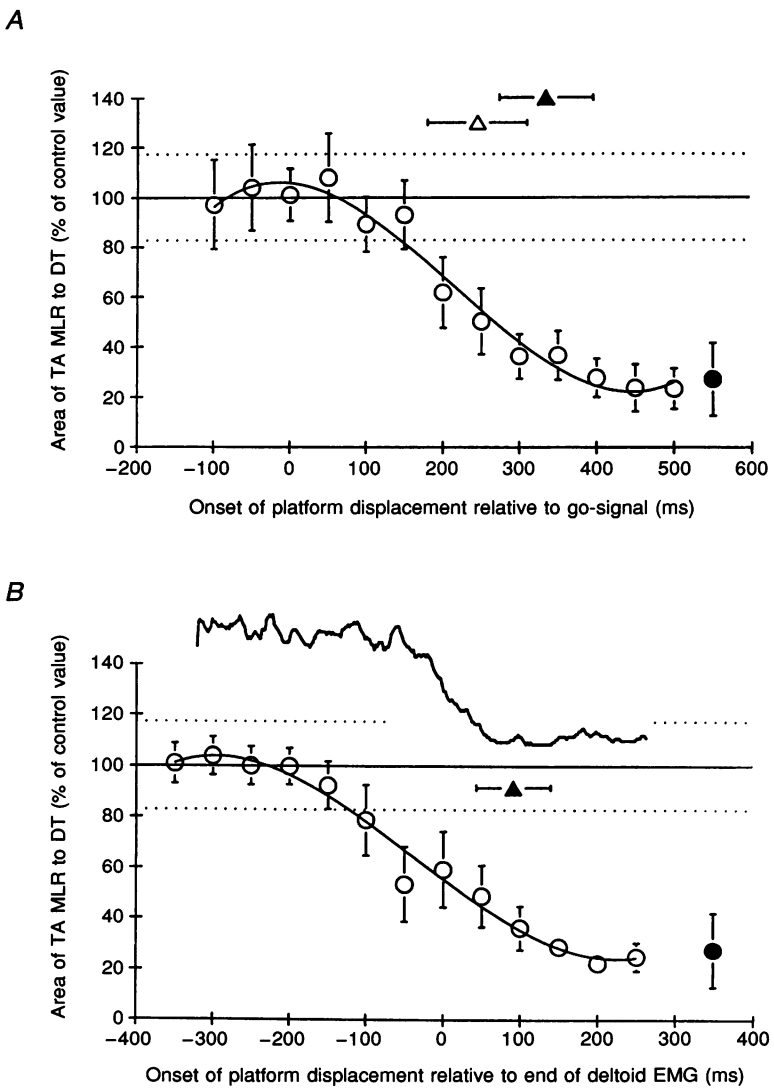

Figure 2. Time course of the average changes in the area of the TA MLR to downward tilt (DT) during the holding task (grand means from all subjects ± S.E.M.)

A, the first significant decrease with respect to control value (± S.D., dotted line) was present when the platform displacement was given 200 ms after the delivery of the visual stimulus, therefore in advance of both the end of deltoid (Delt) tonic activity (△, grand mean ± S.D.) and the subsequent contact between hand and frame (▲, grand mean ± S.D.). ●, the grand mean of the area of the response evoked under steady-holding condition. *B*, same as above, but the time course of the TA MLR was referred to the end of Delt EMG activity. A significant decrease was already present when the platform displacement was induced 100 ms before the end of Delt tonic activity. The profile of the Delt EMG (back-averaged with respect to hand contact) is reported in the graph on the same time scale (zero level of activity corresponds to the horizontal line drawn through the 100% value of the ordinate).

deltoid reference were (a) the time shift to the left of the data points with respect to zero, which reflects the anticipation of EMG termination with respect to hand contact, and (b) the systematically smaller error bars, except in the 'critical' zone. This latter effect most probably depended on the fact that the curves fitted through the values found in the individual subjects were similar in shape, but showed some time shift in their steepest part.

Tibialis anterior LLR

Figure 3 shows the findings for the tibialis anterior LLR, evoked by upward tilt of the platform (see also Fig. 1*A*). The slope of the curves fitted through the data points is similar to that of the curves obtained for the tibialis anterior MLR. The decrease in the response began at about 110 ms from the go-signal (Fig. 3*A*). The response decreased to the values obtained during steady conditions (filled

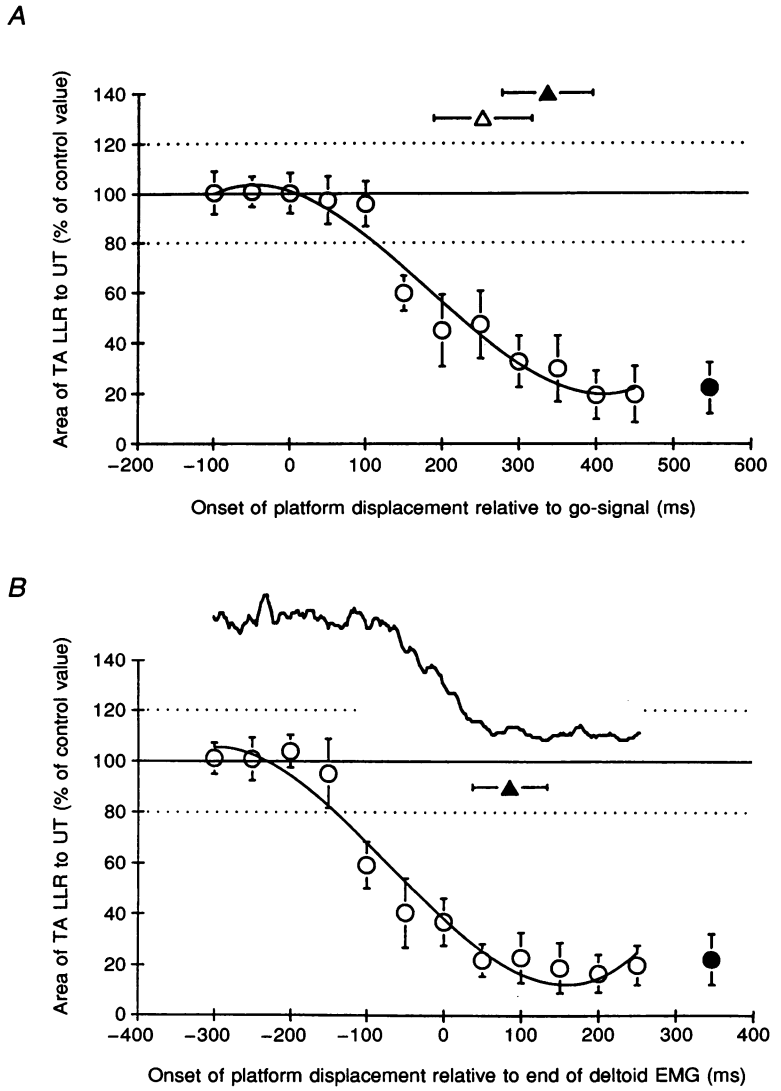

Figure 3. Time course of the changes in the area of the TA LLR to upward tilt (UT) during the holding task (grand means from all subjects ± S.E.M.)

A, the first significant decrease was present when the platform displacement was given 150 ms after the delivery of the visual stimulus, in advance of both the end of Delt tonic activity and the subsequent contact between hand and frame. ●, the grand mean value of the area of the response under steady-holding conditions. *B*, same as above, but the time course of the TA LLR was referred to the end of Delt EMG. A significant decrease was already present when the platform displacement was induced 100 ms before the end of Delt tonic activity. The profile of the Delt EMG decrease is shown in the graph on the same time scale.

circle) within 200 ms from the moment at which the earliest significant reduction was found. In Fig. 3*B*, the plot of the changes in amplitude of the response against the termination of deltoid EMG activity is reported.

Soleus SLR and LLR

Upward tilt and downward tilt of the platform also evoked a short- (SLR) and a long-latency response (LLR), respectively, in the soleus muscle (see Fig. 1*A*). These responses behave differently during stationary holding, since the SLR is not affected, while the LLR is usually reduced in amplitude (Nardone *et al.* 1990*a*; Schieppati & Nardone, 1991). Figure 4 shows that the SLR underwent no significant changes during the holding task (Fig. 4*A*; in both graphs the time reference is the termination of deltoid EMG activity). The soleus LLR significantly decreased during the holding task (Fig. 4*B*), but to a lesser extent than the tibialis LLR described previously. However, the time course over which this response varied from its control value to the value attained during steady holding was not unlike that of the tibialis anterior responses described above.

DISCUSSION

Holding onto a solid bar, thereby stabilizing stance, induces a marked reduction of medium-latency (MLR) and long-latency responses (LLR) of leg muscle to postural perturbations of upright stance. This occurs under stationary conditions, as reported previously, when it was attributed to the new postural 'set' (Nardone *et al.* 1990*a*, *b*;

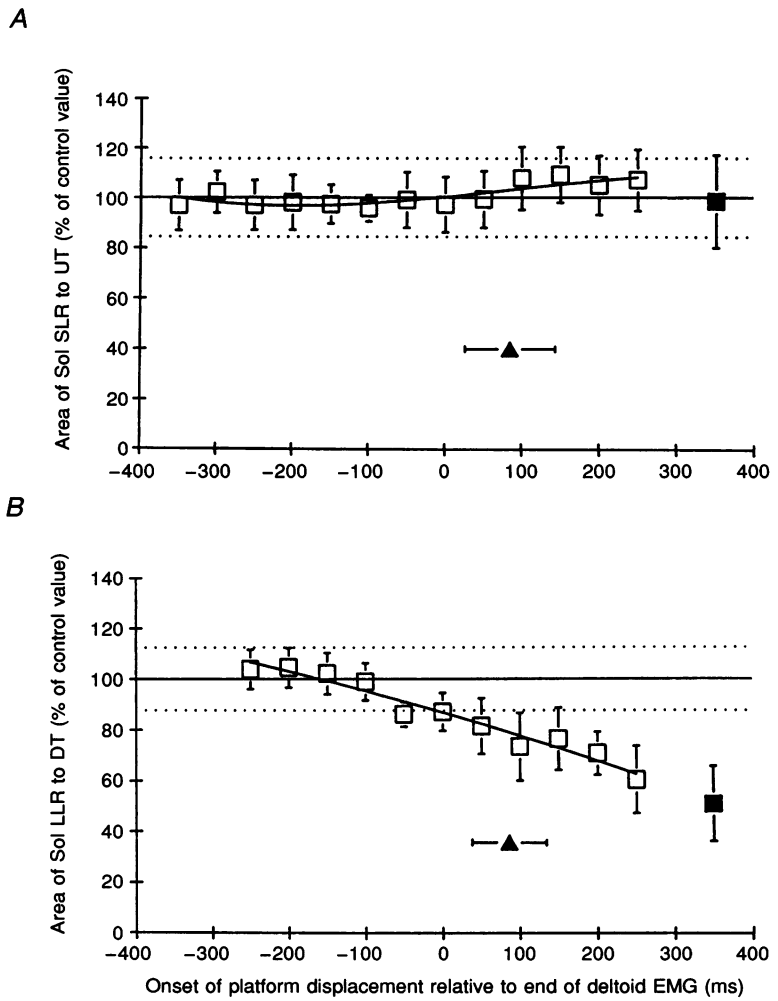

Figure 4. Time course of the changes in the area of the Sol SLR to upward tilt (UT) and Sol LLR to downward tilt (DT) during the holding task (grand means from all subjects ± S.E.M.)

A, the area of the Sol SLR was not significantly affected during the holding task at any interval. ■, grand mean of the area of the response under steady-holding condition. *B*, time course of the decrease in the area of the Sol LLR referred to the end of Delt activity.

Schieppati & Nardone, 1991). It is shown here that the response reduction appears prior to the execution of the holding task, i.e. of the manoeuvre to take hold of the solid frame, and very soon reaches the depression found under stationary conditions. The reduction of the responses before hand contact with the frame rules out the influence on this modulatory phenomenon of cutaneous input from the hand. It also eliminates the possibility that the response reduction induced by stabilization is directly connected to the new 'external reference' (see Massion, 1992), thereby to the new postural set *per se* (for a definition of 'set' see Prochazka, 1989). The short time to reach maximum inhibition points in turn to the operation of a fast, phasic neural mechanism.

Timing of the 'modulatory' effect

The affected responses start to decrease in amplitude to below 1 S.D. of the free stance value when the perturbation was launched between 100 and 150 ms after the visual go-signal that triggered the holding task. However, it should be borne in mind that the tibialis anterior MLR follows the onset of the downward tilt at about 70 ms, whilst the LLRs follows the upward tilt at 120 ms, on average. If these latencies are added to the intervals from the go-stimulus at which the respective responses were decreased (MLR, 140 ms; LLR, 110 ms), one finds that attenuation of both responses begins at about the same latency (about 210–230 ms) from the go-stimulus. If we postulate that the time at which the responses are issued from the spinal cord is shorter than that by about 20 ms (the efferent conduction time, calculated as about half the latency of the soleus SLR, 40 ms), it may be expected with some approximation that the decrease in the excitability of the spinal motoneurones initiates 200 ms after the go-signal. This latency is shorter than the average time from the go-signal at which the deltoid motoneurones end discharging, with a value of 240 ms under our conditions assuming an efferent conduction time of about 5 ms (Ugawa, Genba, Shimpo & Mannen, 1989). The tuning of the responses would even occur ahead of the beginning of deltoid motoneurones derecruitment, if one takes into account the time course of the decrease in deltoid EMG and the more distant site of the lumbar motoneurones with respect to the cervical ones.

Preparatory postural adjustment or gain presetting

The association between the suppression of both medium- and long-latency reflex responses and the cessation of activity of the deltoid motoneurones would indicate a strict time locking between the tuning process and the command leading to stabilization (see also Nardone & Schieppati, 1988). Such co-ordination might imply a feedforward control from the command to perform the focal movement – the holding task – to the centres ultimately responsible for the decrease in excitability of the reflex responses, a situation which occurs during preparatory postural adjustments (Massion, 1992). However, under the conditions deployed in the present study, there was no need to minimize the equilibrium disturbance due to the arm movement, or to provide additional force for the movement itself by shifting the body centre of gravity (not least, because a shift of the centre of gravity would have displaced the hand from its target frame). Further, the association of the onset of response reduction with the termination of deltoid activity was not stronger than with the go-signal, therefore giving no particular support to the notion of a simultaneous control of focal movement and reflex circuits. Finally, the independence of the time course of amplitude modulation from both the latency of the responses (MLR or LLR) and the muscle in which the responses take place suggests that the neural activity responsible for the modulation reaches the spinal cord without a particular spatio-temporal pattern, regardless of the motoneuronal pool (antagonist muscle responses are equally affected) and the type of perturbation (displacing the body centre of gravity either forward or backward). This finding is against a strict posture-stabilizing role of the modulation of the responses.

The changes in the amplitude of the responses with respect to the control value occurred after the delivery of the go-stimulus, thus allowing us to also exclude that gain presetting connected with the expectation of the go-signal, as occurs for instance with the H reflex prior to a reaction time muscle contraction (Schieppati, Nardone & Musazzi, 1986), might have played a role in the modulation of postural reflexes. The notion that the holding condition brought about a decrease in the level of attention, leading to attenuation of reflex gain, can be excluded as well, since the subjects were ready to accomplish the holding task in a reaction-time situation, and because response diminution occurred ahead of the actual contact with the bar.

Therefore, we suggest that the postural response reduction induced by holding simply reflects the needs of the new 'set' that corresponds to the stabilized condition, just as a different 'set' corresponds to locomotion or other states (see Prochazka, 1989). From this perspective, the time course of the effect reflects the time necessary for the new state of spinal reflex excitability to ensue. The adaptive advantage conferred by the new set would simply be that of withholding unnecessary muscle activation, which would have required in turn further effort to keep the body and head still.

Site of action of the modulation

The activity of motoneurones participating in responses with widely different latencies in different destabilizing conditions was suppressed at about the same delay from the go-stimulus. This would be easily explained if the suppressing action were exerted at the level of the motoneurones themselves. However, this hypothesis is readily dismissed by the lack of suppression of the short-latency response (SLR) of soleus, largely monosynaptic (Siliotto *et al.* 1995). Therefore, the suppressing drive must act at a

premotoneuronal level, but the mechanism of action cannot be the enhancement of presynaptic inhibition, otherwise the monosynaptic reflexes would again be depressed (Schieppati, 1987, 1992). During a different 'set' – locomotion – the short-latency stretch reflexes are instead depressed, most probably by presynaptic inhibition (Dietz, Quintern & Berger, 1985; Capaday & Stein, 1986; Llewellyn, Prochazka & Vincent, 1987).

There are strong indications that the MLR of the tibialis anterior is mediated by spindle group II afferent fibres (Dietz, 1992; Siliotto *et al.* 1994; Schieppati, Nardone & Corna, 1995). Studies in cats have shown that reflexes mediated by group II afferent fibres are selectively depressed at the level of their first relay neurone in the spinal cord by monoamines released from monoaminergic systems descending from the brainstem (Bras, Cavallari, Jankowska & McCrea, 1989; see Jankowska, 1993). Similarly, the suppression of the human tibialis anterior MLR could be mediated by the monoaminergic system descending from the brainstem, as evidenced by the effect of tizanidine, an α-2-adrenergic substance, on this response (Schieppati *et al.* 1995; Corna, Grasso, Nardone & Schieppati, 1995). Even the latency and temporal profile of the build-up of the inhibition, and its final stage, appear to be superimposable to the analogous effect observed in cats (Noga, Bras & Jankowska, 1992). Seen in this light, the findings strengthen the hypothesis that MLR is transmitted through the interneurones recipient of group II input from the periphery. The similar time course of suppression of the MLRs and LLRs might in turn imply that also the latter responses are ultimately relayed through the same interneurones.

Incidentally, the brainstem centres of origin of the monoaminergic pathways are both accessed by the output of the motor component of the basal ganglia and affected by Parkinson's disease (Jenner, 1989). Therefore, it is hardly surprising that the depression of the medium- and long-latency responses to postural displacements evoked under stabilized stance in Parkinsonians is either absent or severely impaired (Schieppati & Nardone, 1991).

Armitage, P. (1971). *Statistical Methods in Medical Research.* John Wiley, New York.

Berger, W., Dietz, V. & Horstmann, G. (1988). Are 'long-loop' reflexes involved in the stabilization of posture? *European Journal of Physiology* **411**, R130.

Bras, H., Cavallari, P., Jankowska, E. & McCrea, D. (1989). Comparison of effects of monoamines on transmission in spinal pathways from group I and II muscle afferents in the cat. *Experimental Brain Research* **76**, 27–37.

Capaday, C. & Stein, R. B. (1986). Amplitude modulation of the soleus H-reflex in the human during walking and standing. *Journal of Neuroscience* **6**, 1308–1313.

Corna, S., Grasso, M., Nardone, A. & Schieppati, M. (1995). Selective depression of medium-latency leg and foot muscle responses to stretch by an α_2-agonist in humans. *Journal of Physiology* **483**, 803–809.

Dietz, V. (1992). Human neuronal control of automatic functional movements: interaction between central programs and afferent input. *Physiological Reviews* **72**, 33–69.

Dietz, V., Quintern, J. & Berger, W. (1985). Afferent control of human stance and gait: evidence for blocking of group I afferents during gait. *Experimental Brain Research* **61**, 153–163.

Fellows, S. J., Dömges, F., Töpper, R., Thilmann, A. F. & Noth, J. (1993). Changes in the short- and long-latency stretch reflex components of the triceps surae muscle during ischaemia in man. *Journal of Physiology* **472**, 737–748.

Huttunen, J. & Hömberg, V. (1990). EMG responses in leg muscles to postural perturbations in Huntington's disease. *Journal of Neurology, Neurosurgery and Psychiatry* **53**, 55–62.

Jankowska, E. (1993). Monoaminergic inhibitory control of spinal interneurons. In *Spasticity: Mechanisms and Management*, ed. Thilmann, A. F., Burke, D. J. & Rymer, W. Z., pp. 222–232. Springer-Verlag, Berlin.

Jenner, P. (1989). MPTP-induced Parkinsonism: the relevance to idiopathic Parkinson's disease. In *Disorders of Movement: Clinical, Pharmacological and Physiological Aspects*, ed. Quinn, N. P. & Jenner, P. G., pp. 157–176. Academic Press, London.

Llewellyn, M., Prochazka, A. & Vincent, S. (1987). Transmission of human tendon jerk reflexes during stance and gait. *Journal of Physiology* **382**, 82*P*.

Massion, J. (1992). Movement, posture and equilibrium: interaction and coordination. *Progress in Neurobiology* **38**, 35–56.

Nardone, A., Corrà, T. & Schieppati, M. (1990*a*). Different activations of the soleus and gastrocnemii muscles in response to various types of stance perturbation in man. *Experimental Brain Research* **80**, 323–332.

Nardone, A., Giordano, A., Corrà, T. & Schieppati, M. (1990*b*). Responses of leg muscles in humans displaced while standing. Effects of types of perturbation and of postural set. *Brain* **113**, 65–84.

Nardone, A. & Schieppati, M. (1988). Postural adjustments associated with voluntary contraction of leg muscles in standing man. *Experimental Brain Research* **69**, 469–480.

Noga, B. R., Bras, H. & Jankowska, E. (1992). Transmission from group II muscle afferents is depressed by stimulation of locus coeruleus/subcoeruleus, Kölliker–Fuse and raphe nuclei in the cat. *Experimental Brain Research* **88**, 502–516.

Prochazka, A. (1989). Sensorimotor gain control: a basic strategy of motor systems? *Progress in Neurobiology* **33**, 281–307.

Schieppati, M. (1987). The Hoffmann reflex: a means of assessing spinal reflex excitability and its descending control in man. *Progress in Neurobiology* **28**, 345–376.

Schieppati, M. (1991). Motor unit reflex activity, normal and pathological. *Current Opinion in Neurology and Neurosurgery* **4**, 749–755.

Schieppati, M. (1992). Selection of task-related motor output through spinal interneurones. *Behavioral and Brain Sciences* **15**, 794–795.

Schieppati, M. & Nardone, A. (1991). Free and supported stance in Parkinson's disease. The effect of posture and postural set on leg muscle responses to perturbation, and its relation to the severity of the disease. *Brain* **114**, 1227–1244.

SCHIEPPATI, M., NARDONE, A. & CORNA, S. (1995). Do secondary spindle afferent fibres play a role in the late response to stretch of leg muscles in humans? In *Alpha and Gamma Motor Systems*, ed. TAYLOR, A., GLADDEN, M. H. & DURBABA, R. Plenum Publishing Corporation, New York (in the Press).

SCHIEPPATI, M., NARDONE, A., GRASSO, M. & SILIOTTO, R. (1993). Time-course of amplitude changes of leg muscle responses to stance perturbation during a postural stabilization task in humans. *XXXII Congress of the International Union of Physiological Sciences* 170.33/P.

SCHIEPPATI, M., NARDONE, A. & MUSAZZI, M. (1986). Modulation of the Hoffmann reflex by rapid muscle contraction or release. *Human Neurobiology* **5**, 59–66.

SCHOLZ, E., DICHGANS, J., GUSCHLBAUER, B. & ACKERMANN, H. (1990). What are the afferents to the 'long loop' reflexes? In *Disorders of Posture and Gait*, ed. BRANDT, T., PAULUS, W., BLES, W., DIETERICH, M., KRAFCZYK, S. & STRAUBE, A., pp. 94–97. Georg Thieme Verlag, Stuttgart.

SILIOTTO, R., GRASSO, M., NARDONE, A. & SCHIEPPATI, M. (1995). Contribution of foot muscle responses to perturbation of stance. In *Motor Control VII*, ed. STUART, D. G., GANTCHEV, G. N., GURFINKEL, V. S. & WIESENDANGER, M. Motor Control Press, Tucson, AZ, USA (in the Press).

UGAWA, Y., GENBA, K., SHIMPO, T. & MANNEN, T. (1989). Physiologic analysis of central motor pathways – Simultaneous recording from multiple relaxed muscles. *European Neurology* **29**, 135–140.

Acknowledgements

We wish to thank Gillian Jarvis for English language editing and Margherita Grasso for her assistance in the experiments. This study was supported in part by the grant 'Ricerca Finalizzata 1994/97' from the Italian Ministry of Health.

Received 10 November 1994; accepted 15 March 1995.